

AMY BRYANT, M.D., M.S.C.R.; BEVERLY  
GRAY, M.D., ELIZABETH DEANS, M.D.,  
on behalf of themselves and their patients  
seeking abortions; and PLANNED  
PARENTHOOD SOUTH ATLANTIC, on  
behalf of itself, its staff and its patients  
seeking abortions,  
  
Plaintiffs,  
  
v.  
  
JIM WOODALL, in his official capacity as  
District Attorney ("DA") for Prosecutorial  
District ("PD") 15B; Roger Echols, in his  
official capacity as DA for PD 14; Eleanor  
E. Greene, M.D., M.P.H., in her official  
capacity as Secretary of the North Carolina  
Medical Board; and Rick Brajer, in his  
official capacity as Secretary of the North  
Carolina Department of Health and Human  
Services; and their employees, agents, and  
successors,  
  
Defendants.

CIVIL ACTION NO.  
1:16-cv-01368-UA-LPA

**DECLARATION OF  
MAUREEN L. CONDON  
Ph.D**

2. I am an Associate Professor of Neurobiology and Anatomy at the University of Utah School of Medicine, with an adjunct appointment in the

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**DECLARATION OF  
MAUREEN L. CONDIE,  
Ph.D**

2. I am an Associate Professor of Neurobiology and Anatomy at the University of Utah School of Medicine, with an adjunct appointment in the

Department of Pediatrics. I received my undergraduate degree from the University of Chicago and my doctorate from the University of California at Berkeley.

3. Since my appointment at the University of Utah in 1997, my primary research focus has been the development and regeneration of the nervous system, focused on the role of neural stem cells. In 1999, I was awarded the Basil O'Connor Young Investigator Award for my studies of peripheral nervous system development. In 2002, I was named a McKnight Neuroscience of Brain Disorders Investigator in recognition of my research in the field of adult spinal cord regeneration.. My current research involves the molecular genetics of human pluripotent stem cells.

4. In addition to my scientific research, I participate in both graduate and medical teaching. I am the director of the University of Utah School of Medicine curriculum in Human Embryology. I have published and presented seminars nationally and internationally on issues concerning human embryology, science policy and the ethics of biological research. A true and correct copy of my current *curriculum vitae* is attached to this Declaration as Exhibit A.

5. The purpose of this Declaration is to state my opinion, based on the extant medical and scientific data, on the question whether a human fetus can feel pain and, if so, at what stage of fetal development a human fetus can feel pain.

6. The scientific evidence regarding the development of human brain structures demonstrates that, by 18-weeks post sperm/egg fusion (20 weeks, as dated from the last menstrual period), a human fetus is capable of detecting and responding to pain.

7. In the simplest sense, pain is an aversive response to a “noxious” (that is, physically harmful or destructive) stimulus. The medical dictionary administered by the National Institutes of Health (the “NIH”)<sup>1</sup> supports this view, defining pain as “a basic bodily sensation that is induced by a noxious stimulus, is received by naked nerve endings, is characterized by physical discomfort (such as pricking, throbbing, or aching), and typically leads to evasive action.”

8. Nevertheless, pain has more complex dimensions. The NIH dictionary also offers the following, more nuanced, definition of pain: “a state of physical, emotional, or mental lack of wellbeing or physical, emotional, or mental uneasiness that ranges from mild discomfort or dull distress to acute, often unbearable agony, may be generalized or localized, and is the consequence of being injured or hurt.” This definition also indicates that pain is a response to a noxious stimulus or injury, but acknowledges that the response can have emotional or mental dimensions as well. And, like all mental experiences, it is difficult for any one of us to fully appreciate another person’s psychological experience of pain.

9. The scientific evidence regarding the development of pain circuitry is entirely undisputed and has been reported in every modern review of fetal pain, with essentially the same interpretation as is given below.<sup>2</sup>

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<sup>1</sup> See definition “b” at: <http://www.merriam-webster.com/medlineplus/pain>.

<sup>2</sup> See, for example: Is Fetal Pain a Real Evidence?, Bellieni, C.V., Buonocore, G. J., *Matern. Fetal Neonatal Med.*, 2012 Aug; 25(8):1203-8; *Neuro Endocrinol Lett.*, 2008; Dec; 29(6):807-14; *Neurodevelopmental Changes of Fetal Pain*, Lowery, C.L., Hardman, M.P., Manning, N., Hall, R.W., Anand, K.J., Clancy, B., *Semin. Perinatol.*, 2007 Oct; 31(5):275-82; *Fetal Pain Perception and Pain Management*, Van de Velde,

10. When does the fetus develop neural circuitry capable of pain perception?

The ability to perceive noxious stimuli and react to them develops over a very long period of time in humans, continuing well after birth. The earliest “rudiment” of the human nervous system forms by 28 days (*i.e.*, four weeks).<sup>3</sup> At this stage, the primitive brain is already “patterned,” *i.e.*, cells in different regions are specified to produce structures appropriate to their location and function in the nervous system as a whole.<sup>4</sup> The brain grows enormously over the next several weeks, such that, by 50 days, the rudiments of the major regions of the central nervous system have all been established.

11. In the region of the brain responsible for thinking, memory and other “higher” functions, the earliest neurons are generated during the fourth week.<sup>5</sup> In

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M., Jani, J., De Buck, F., Deprest, J., Semin. Fetal Neonatal Med., 2006 Aug; 11(4):232-6. Epub 2006 Apr. 18; Fetal Pain: A Systematic Multidisciplinary Review of the Evidence, Lee, S.J., Ralston, H.J., Drey, E.A., Partridge, J.C., Rosen, M.A., JAMA. 2005 Aug. 24; 294(8):947-54; The Development of Nociceptive Circuits, Fitzgerald, M., Nat. Rev. Neurosci., 2005 Jul; 6(7):507-20.

<sup>3</sup> Throughout this discussion, the age of the fetus is given in weeks post sperm-egg fusion. For the equivalent “gestational age,” or week of pregnancy dated from last menstrual period, two weeks must be added to the ages provided. When quoting papers that refer to “gestational age,” the age of the fetus has been reported in square brackets to remain consistent throughout the discussion.

<sup>4</sup> Langman’s Medical Embryology, 11th Ed., Sadler, T.W. (2009), Lippincott Williams and Wilkins. (ISBN-10: 0781790697) Chapters 5 and 6.

<sup>5</sup> Tangential Networks of Precocious Neurons and Early Axonal Outgrowth in the Embryonic Human Forebrain, Bystron, I., Molnár, Z., Otellin, V., Blakemore, C. J., Neurosci., 2005; 25:2781-92; ApoER2 and VLDLR in the Developing Human Telencephalon, Cheng, L., Tian, Z., Sun, R., Wang, Z., Shen, J., Shan, Z., Jin, L., Lei, L., Eur. J., Paediatr. Neurol., 2011; 15:361-7; The First Neurons of the Human Cerebral Cortex, Bystron, I., Rakic, P., Molnár, Z., Blakemore, C., Nat. Neurosci.,

animals, synapses that allow for communication between cortical neurons are functional immediately and it is likely that this is also true of humans.

12. The neural circuitry responsible for the most primitive response to pain, the spinal reflex, is in place by eight weeks. This is the earliest point at which the fetus is capable of detecting and reacting to painful stimuli in any capacity.<sup>6</sup> And a fetus responds just as humans at later stages of development respond: by actively withdrawing from the painful stimulus.

13. The earliest connections between neurons in the subcortical-frontal pathways (the regions of the brain involved in motor control and a wide range of psychological phenomena, including pain perception) are detected by 37 days and are well established by 8-10 weeks.<sup>7</sup> Components of these circuits include the basal ganglia, limbic system, thalamus and hypothalamus.

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2006; 9:880-6. Epub 2006 June 18.; Development of the Human Cerebral Cortex: Boulder Committee Revisited, Bystron, I., Blakemore, C., Rakic, P., Nat. Rev. Neurosci., 2008; 9:110-22.

<sup>6</sup> Synaptogenesis in the Cervical Cord of the Human Embryo: Sequence of Synapse Formation in a Spinal Reflex Pathway, Okado, N., Kakimi, S., Kojima, T. J., Comp. Neurol., 1979; 184:491-518; Onset of Synapse Formation in the Human Spinal Cord, Okado, N., J. Comp. Neurol., 1981; 201:211-9; The Fine Structure of the Spinal Cord in Human Embryos and Early Fetuses, Wozniak, W., O'Rahilly, R., Olszewska, B. J. Hirnforsch, 1980; 21:101-24; Early Synaptogenesis in the Spinal Cord of Human Embryos, Milokhin, A.A., Acta, Biol. Hung. 1983; 34:231-45; Development of Pain Mechanisms, Fitzgerald, M., Br. Med Bull. 1991; 47:667-75.

<sup>7</sup> Development of Axonal Pathways in the Human Fetal Fronto-Limbic Brain: Histochemical Characterization and Diffusion Tensor Imaging, Vasung, L., Huang, H., Jovanov-Milošević, N., Pletikos, M., Mori S., Kostović I., J. Anat., 2010; 217:400-17; Insights From In Vitro Fetal Magnetic Resonance Imaging of Cerebral Development, Kostovic, I., Vasung, L., Semin, Perinatol.2009; 33:220-33.

14. Connections between the spinal cord and subcortical structures in the thalamus, a region of the brain that is critically involved in pain perception at all developmental stages, begin to form around 12 weeks<sup>8</sup> and are completed by 18 weeks.<sup>9</sup>

15. Thalamo-cortical connections and long-range connections within the cortex do not arise until later in fetal life, beginning around 22-24 weeks,<sup>10</sup> and continuing to develop for an exceptionally long time, not reaching full maturity until approximately 25 years after birth.<sup>11</sup>

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<sup>8</sup> Transient Cholinesterase Staining in the Mediodorsal Nucleus of the Thalamus and Its Connections in the Developing Human and Monkey Brain, Kostovic, I., Goldman-Rakic, P.S., J. Comp. Neurol., 219:431-447, 1983.

<sup>9</sup> Transient Cholinesterase Staining in the Mediodorsal Nucleus of the Thalamus and Its Connections In the Developing Human and Monkey Brain, Kostovic, I., Goldman-Rakic, P.S., J. Comp. Neurol., 219:431-447, 1983.

<sup>10</sup> Functional Maturation of Neocortex: A Base of Viability, Gatti, M.G., Becucci, E., Fagioli, F., Fagioli, M., Ádén, U., Buonocore, G. J., Matern. Fetal Neonatal Med., 2012; 25 Suppl. 1:101-3; 3D Global and Regional Patterns of Human Fetal Subplate Growth Determined in Utero, Corbett-Detig, J., Habas, P.A., Scott, J.A., Kim, K., Rajagopalan, V., McQuillen, P.S., Barkovich, A.J., Glenn, O.A., Studholme, C., Brain Struct. Funct., 2011; 215:255-63.; The Development of the Subplate and Thalamocortical Connections in the Human Fetal Brain, Kostović, I., Judas, M., Acta Paediatr., 2010 Aug; 99(8):1119-27.

<sup>11</sup> Dynamic Mapping of Human Cortical Development During Childhood Through Early Adulthood, Gogtay, N., et al., Proc. Nat'l Acad. Sci. USA 2004; 101:8174; Sowell, E.R., et al. (2003), Mapping Cortical Change Across the Human Life Span, Nat. Neurosci., 6:309; Changes of Brain Activity in the Neural Substrates For Theory of Mind During Childhood and Adolescence, Moriguchi, Y., Ohnishi, T., Mori, T., Matsuda, H., Komaki, G., Psychiatry Clin. Neurosci. 2007 Aug; 61(4):355-63.

16. It is generally accepted that the simplest neural circuitry required to detect and respond to pain is in place by 8-10 weeks of human development.<sup>12</sup> What is not universally accepted is at what point in development the ability to detect and respond to pain becomes psychologically and emotionally meaningful—both to the fetus and to society. Stated another way, the debate over fetal pain is not whether a fetus detects pain in some manner during the first trimester of life (all parties agree on this point), but rather how pain is experienced, *i.e.*, whether a fetus is capable of “suffering.”

17. Two major reviews of the scientific literature on fetal pain are commonly cited on this topic; one published by the Royal College of Obstetricians and Gynecologists (hereafter the “RCOG”) in 2010,<sup>13</sup> and one published in the Journal of the American Medical Association (hereafter referred to as “JAMA”) in 2005.<sup>14</sup> Both discuss the same scientific literature presented here and both agree that the early arising spinal circuits sufficient for detection and response to pain (*i.e.*, “nociception”) are in place by 8-10 weeks, with more sophisticated thalamic pain circuitry in place by 18 weeks.

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<sup>12</sup> *Ibid* at 6.

<sup>13</sup> RCOG, Fetal Awareness: Review of Research and Recommendations for Practice (Mar. 2010). Available here: <https://www.rcog.org.uk/globalassets/documents/guidelines/rcogfetalawarenesswpr0610.pdf>

<sup>14</sup> Fetal Pain: A Systematic Multidisciplinary Review of the Evidence, Lee, S.J., Ralston, H.J., Drey, E.A., Partridge, J.C., Rosen, M.A., JAMA. 2005; 294:947-54.

18. Yet despite acknowledging the scientific facts regarding early fetal pain perception, both reviews somewhat paradoxically conclude that the fetus does not experience pain in a meaningful sense during the first two trimesters, because, they insist, late developing cortical circuitry is required for the conscious experience of pain that we call “suffering.” A position statement by the American College of Obstetricians and Gynecologists (hereafter the “ACOG”) issued in 2012<sup>15</sup> draws the same perplexing conclusion (*i.e.*, that, prior to 30 weeks, a fetus does not have the neural circuitry required for consciousness and “suffering”), based largely on the RCOG review.

19. In considering this paradox, it is important to note that the RCOG and the ACOG represent the primary providers of abortion services both in the United Kingdom and the United States, and therefore the views of these organizations are likely to entail a significant conflict of interest. Similarly, in 2005, at least two of the five authors of the JAMA review were directly involved in providing abortion services.<sup>16</sup>

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<sup>15</sup> Amicus Curiae Brief, the ACOG and the American Congress of Obstetricians and Gynecologists. *Isacson v. Horne*, No. 12-16670, United States Court of Appeals for the Ninth Circuit. Available here: [http://cdn.ca9.uscourts.gov/datastore/general/2012/09/26/12\\_16670\\_Amicus\\_Brief.pdf](http://cdn.ca9.uscourts.gov/datastore/general/2012/09/26/12_16670_Amicus_Brief.pdf)

<sup>16</sup> The biographical information for Eleanor Drey, M.D., Ed.M. (available at: <http://bixbycenter.ucsf.edu/fs/bios/drey-eleanor.html>) states that she is “Medical Director of the Women’s Options Center of San Francisco General Hospital and an Associate Clinical Professor in the Department of Obstetrics, Gynecology and Reproductive Sciences of the University of California, San Francisco.” The C.V. of Mark A. Rosen, M.D. (available at:

20. In light of this potential bias, it is important to ask: What scientific evidence is presented by the RCOG and the JAMA in support of the surprising and pivotal assertion that, although an 18-week fetus clearly possesses the neural circuitry required to detect and respond to pain, this response is irrelevant, because late-developing cortical circuitry is required for both consciousness and for “suffering?” Remarkably, the answer to this question is “none,” *i.e.* the RCOG and the JAMA present absolutely no data in support of this critical claim.

21. The RCOG review boldly states that: “Most pain neuroscientists believe that the cortex is necessary for pain perception,”<sup>17</sup> yet cites only three papers in support of this key assertion, one study of resting-state brain activity in infants that does not address pain perception,<sup>18</sup> one study in adults that, in direct contradiction of the RCOG’s claims, indicates multiple non-cortical regions are involved in pain perception (the hypothalamus, periaqueductal grey matter and the thalamus),<sup>19</sup> and a third study, also in adults, that addresses pain sensitivity but does not directly

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<http://anessom.ucsd.edu/VP%20Articles/RosenCV.pdf> indicates his specialty is obstetrical anesthesiology.

<sup>17</sup> RCOG, pg. 11.

<sup>18</sup> Resting-State Networks in the Infant Brain, Fransson, P., Skiöld, B., Horsch, S., Nordell, A., Blennow, M., Lagercrantz, H., Aden, U. Proc. Nat’l Acad. Sci. U S A. 2007 Sep 25; 104(39):15531-6.

<sup>19</sup> The Brain-Heart Axis in the Perception of Cardiac Pain: the Elusive Link Between Ischaemia and Pain, Rosen, S.D., Camici, P.G., Ann. Med. 2000 Jul; 32(5):350-64.

determine what brain structures are required for either consciousness or “suffering.”<sup>20</sup>

22. Similarly, the JAMA review flatly asserts that: “Pain is a subjective sensory and emotional experience that requires the presence of consciousness.”<sup>21</sup> Yet like the RCOG, the JAMA also does not list a single scientific study in support of this far-reaching claim. Instead, the JAMA refers the reader to a website maintained (at that time) by the International Society for the Study of Pain as a resource for terminology and cites a pair of expert opinion papers, neither of which directly addresses the role of the cortex in either consciousness or pain perception. Thus neither the JAMA nor the RCOG presents a single piece of scientific evidence that a fetus is incapable of consciousness or that “suffering” requires cortical circuitry.

23. Is there a “consensus” on development of pain perception in human fetuses? The JAMA review is the most widely cited authority on the topic of fetal pain, and is often said to reflect the “consensus” of expert opinion. Yet it is important to ask: do the JAMA’s conclusions accurately represent the views of most neuroscientists—even at the time? Multiple lines of evidence clearly contradict the view presented by the JAMA (see paragraphs 26-39 below). Yet surprisingly, even the expert opinion papers cited by the JAMA itself (papers that also review the pain

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<sup>20</sup> Neural Correlates of Interindividual Differences in the Subjective Experience of Pain, Coghill, R.C., McHaffie, J.G., Yen, Y.F., Proc. Nat’l Acad. Sci. U S A. 2003 Jul 8;100(14):8538-42.

<sup>21</sup> JAMA, pg. 848.

literature available at the time) do not agree with the JAMA's interpretation of the evidence.

24. The first of these papers disagrees with the JAMA's conclusion that "tests of cortical function suggest that conscious perception of pain does not begin before [29 or 30 weeks fetal age],"<sup>22</sup> instead concluding that the available evidence suggests pain perception commences considerably earlier, stating, "fetuses of around [26-28 weeks fetal age] are capable of feeling pain."<sup>23</sup> (The authors of this expert review do not address the experience of younger fetuses, likely due to the lack of sufficient evidence at the time, but see the evidence presented on this subject in paragraphs 26-39 below.)

25. The second paper cited by the JAMA also rejects the conclusion that a fetus is incapable of suffering before 29-30 weeks, stating that: "The physical system for nociception is present and functional by [24 weeks fetal age] and it seems likely that the fetus is capable of feeling pain from this stage."<sup>24</sup>

26. Thus, even back in 2005, credible experts were not unanimous in their interpretation of the evidence. The JAMA itself refrains from drawing a firm conclusion, conceding that the "limited" scientific evidence merely indicates "fetal

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<sup>22</sup> JAMA, pg. 952.

<sup>23</sup> A Pain in the Fetus: Toward Ending Confusion About Fetal Pain, Benatar, D., Benatar, M., Bioethics, 2001 Feb; 15(1):57-76.

<sup>24</sup> Fetal Pain: Implications for Research and Practice, Glover, V., Fisk, N.M., Br. J. Obstet. Gynaecol, 1999 Sep; 106(9):881-6.

perception of pain is *unlikely* before the third trimester” (emphasis added; p. 947). And due to the personal nature of pain, uncertainty regarding the psychological experience of the fetus will undoubtedly persist. It may never be possible to scientifically determine whether a fetus is capable of “suffering.”

27. What brain structures are actually required for pain perception? In contrast to the lack of scientific evidence supporting the pivotal assertion of the RCOG and the JAMA that the fetus is incapable of suffering because the cortex is necessary for conscious pain experience, there is an enormous body of scientific data that clearly indicates the cortex is not required for either consciousness or suffering—data that the RCOG and the JAMA simply ignore. Nine independent lines of scientific evidence that strongly contradict the conclusions of the RCOG and the JAMA are summarized briefly below.

28. Although the neocortex is unique to mammals,<sup>25</sup> animals that entirely lack this region of the brain (*e.g.*, fish, amphibians, reptiles and birds) are clearly both conscious and capable of suffering.<sup>26</sup> More recently, extensive studies have determined that the neural structures underlying the most primitive form of

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<sup>25</sup> Genetic and Developmental Homology in Amniote Brains, Toward Conciliating Radical Views of Brain Evolution, Aboitiz, F., Brain Res. Bull., 2011 Feb 1; 84(2):125-36.; Evolution of the Amniote Pallium and the Origins of Mammalian Neocortex, Butler, A.B., Reiner, A., Karten, H.J., Ann. N. Y. Acad. Sci. 2011 Apr; 1225:14-27.

<sup>26</sup> The area of consciousness research is both complex and contested; however, it is obvious that an alert and active animal (*i.e.* a “conscious” animal) is different from an anesthetized or sleeping animal (*i.e.* an “unconscious” animal). Whether the consciousness of an alert animal is the same as that of a human is open to interpretation, but alert animals are clearly capable of suffering.

consciousness in both humans and animals are found in subcortical regions of the brain,<sup>27</sup> with one expert stating categorically: “it is now eminently clear that affective consciousness is a property of subcortical circuits we share with the other animals.”<sup>28</sup> These “subcortical circuits” would include brain structures that are well developed in a human fetus by 18 weeks.

29. Mammals (including rodents, cats and primates) that have had the cortex partially or fully removed remain conscious and continue to show a vigorous response to painful stimuli.<sup>29</sup>

30. Similarly, human children born without the cortex (‘decorticate’ or hydranencephalic patients) are capable of conscious behaviors, including smiling, distinguishing between familiar/unfamiliar people and situations, having preferences

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<sup>27</sup> Reviewed in: Evolutionary Aspects of Self- and World Consciousness in Vertebrates, Fabbro, F., Aglioti, S.M., Bergamasco, M., Clarici, A., Panksepp, J., *Front. Hum. Neurosci.*, 2015 Mar 26; 9:157; Hallmarks of Consciousness, Butler, A.B., *Adv. Exp. Med. Biol.*, 2012; 739:291-309; Animal Consciousness: a Synthetic Approach, Edelman, D.B., Seth, A.K., *Trends Neurosci.* 2009 Sep; 32(9):476-84; 17, Emotion and Cortical-Subcortical Function: Conceptual Developments, Bennett, M.R., Hacker, P.M., *Prog. Neurobiol.*, 2005 Jan; 75(1):29-52.

<sup>28</sup> Cross-Species Affective Neuroscience Decoding of the Primal Affective Experiences of Humans and Related Animals, Panksepp, J., *PLoS One*, 2011; 6(9):e21236.

<sup>29</sup> Effects of Partial Decortication on Opioid Analgesia in the Formalin Test, Matthies, B.K., Franklin, K.B., *Behav. Brain Res.*, 1995; 67:59-66; Formalin pain is expressed in decerebrate rats, but not attenuated by morphine. Matthies, B.K., Franklin, K.B., *Pain*, 1992; 51:199-206; Effects of Selective Prefrontal Decortication on Escape Behavior in the Monkey, Tanaka, D., Jr., *Brain Res.*, 1973 Apr 13; 53(1):161-73; Somatosensory Cortical Involvement in Response to Noxious Stimulation in the Cat., Berkley, K.J., Parmer, R., *Exp. Brain Res.*, 1974; 20(4):363-74.

for particular kinds of music and having adverse reactions to pain.<sup>30</sup> This evidence clearly indicates (in direct contradiction to the unsupported claims of the RCOG and the JAMA) that long-range cortical connections developing only after 22 weeks in the human fetus, and completely absent in these patients, are not necessary for consciousness or for a psychological perception of suffering.

31. Conversely, the largest study conducted to date of human patients with disorders of consciousness<sup>31</sup> unambiguously concludes that loss of subcortical, not cortical, circuitry is associated with loss of consciousness, stating, “clinical measures of awareness and wakefulness upon which differential diagnosis rely were systematically associated with tissue atrophy within thalamic and basal ganglia nuclei.” Moreover, experts in the study of consciousness conclude that consciousness clearly persists in the absence of “vast regions of the cortex.”<sup>32</sup>

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<sup>30</sup> Consciousness Without Cortex: a Hydranencephaly Family Survey, Aleman, B., Merker, B., *Acta Paediatr.* 2014 Oct; 103(10):1057-65; The Presence of Consciousness in the Absence of the Cerebral Cortex, Beshkar, M., *Synapse*, 2008; 62:553-6; Consciousness in Congenitally Decorticate Children: Developmental Vegetative State as Self-Fulfilling Prophecy, Shewmon, D.A., Holmes, G.L., Byrne, P.A., *Dev. Med. Child Neurol.*, 1999; 41:364-74; The Role of Primordial Emotions in the Evolutionary Origin of Consciousness, Denton, D.A., McKinley, M.J., Farrell, M., Egan, G.F., *Conscious Cogn.*, 2009; 18:500-14; Consciousness Without a Cerebral Cortex: a Challenge For Neuroscience and Medicine, Merker, B., *Behav. Brain Sci.*, 2007; 30(1):63-81.

<sup>31</sup> Thalamic and Extrathalamic Mechanisms of Consciousness After Severe Brain Injury, Lutkenhoff, E.S., Chiang, J., Tshibanda, L., Kamau, E., Kirsch, M., Pickard, J.D., Laureys, S., Owen, A.M., Monti, M.M., *Ann. Neurol.* 2015 Jul; 78(1):68-76.

<sup>32</sup> Minimal Neuroanatomy for a Conscious Brain: Homing in on the Networks Constituting Consciousness, Morsella, E., Krieger, S.C., Bargh, J.A., *Neural Netw.* 2010; 23:14-5.

32. Recent authoritative reviews of the neural basis of consciousness and emotion in humans also do not support the claims of the RCOG and the JAMA that conscious feelings (including suffering) are exclusively represented in the cortex. Rather, these experts conclude that “the available evidence indicates that phylogenetically recent sectors of the nervous system, such as the cerebral cortex, contribute to but are not essential for the emergence of feelings, which are likely to arise instead from older regions such as the brainstem” and that the “neural substrates [of consciousness] can be found at all levels of the nervous system.”<sup>33</sup>

33. Although anesthesia has been used for over 150 years, the precise mechanisms by which anesthetics suppress both consciousness and pain are not well understood.<sup>34</sup> However, recent work using high resolution brain imaging in both

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<sup>33</sup> The Nature of Feelings: Evolutionary and Neurobiological Origins, Damasio, A., Carvalho, G.B., *Nat. Rev. Neurosci.*, 2013;14:143-52.

<sup>34</sup> Cerebral Mechanisms of General Anesthesia, Uhrig, L., Dehaene, S., Jarraya, B., *Ann. Fr. Anesth. Reanim.* 2014 Feb; 33(2):72-82. doi: 10.1016/j.annfar.2013.11.005. Epub 2013 Dec 22; General Anaesthesia: From Molecular Targets to Neuronal Pathways of Sleep and Arousal, Franks, N.P., *Nat. Rev. Neurosci.*, 2008 May; 9(5):370-86; Mechanisms of Anesthesia: Towards Integrating Network, Cellular, and Molecular Level Modeling, Arhem, P., Klement, G., Nilsson, J., *Neuropsychopharmacology*, 2003 Jul; 28 Suppl 1:S40-7.

animals<sup>35</sup> and humans<sup>36</sup> strongly indicates that anesthesia-induced loss of consciousness is associated with a reduction in the activity of the thalamus that is only later followed by suppression of cortical activity in response to reduced thalamic function. These studies indicate that consciousness—and therefore conscious pain perception—depends on thalamic, not cortical, circuitry. And thalamic circuitry is well developed in the human fetus by 18 weeks.

34. The cortical regions associated with processing of painful experiences (dorsal-lateral prefrontal cortex and dorsal-anterior cingulate cortex<sup>37</sup>) continue to develop for decades after birth, with these regions being among the last to achieve maturity.<sup>38</sup> However, our perception of physical pain and suffering remains relatively

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<sup>35</sup> Attenuation of high-frequency (50-200 Hz) thalamocortical EEG rhythms by propofol in rats is more pronounced for the thalamus than for the cortex. Reed, S.J., Plourde, G., PLoS One, 2015 Apr 15; 10(4):e0123287; Altered activity in the central medial thalamus precedes changes in the neocortex during transitions into both sleep and propofol anesthesia. Baker, R., Gent, T.C., Yang, Q., Parker S., Vyssotski, A.L., Wisden, W., Brickley, S.G., Franks, N.P., J. Neurosci., 2014 Oct 1; 34(40):13326-35.

<sup>36</sup> Anesthetic Effects of Propofol in the Healthy Human Brain: Functional Imaging Evidence, Song, X.X., Yu, B.W., J. Anesth. 2015 Apr; 29(2):279-88. The thalamus and brainstem act as key hubs in alterations of human brain network connectivity induced by mild propofol sedation. Gili, T., Saxena, N., Diukova, A., Murphy, K., Hallm, J.E., Wisem, R.G., J. Neurosci., 2013 Feb 27; 33(9):4024-31. Thalamus, brainstem and salience network connectivity changes during propofol-induced sedation and unconsciousness. Guldenmund, P., Demertzim, A., Boverouxm, P., Bolym, M., Vanhaudenhuyse, A., Bruno, M.A., Gosseries, O., Noirhomme, Q., Brichant, J.F., Bonhomme, V., Laureys, S., Soddu, A., Brain Connect, 2013; 3(3):273-85.

<sup>37</sup> Imaging CNS Modulation of Pain in Humans, Bingel, U., Tracey, I., Physiology (Bethesda). 2008 Dec; 23:371-80.

<sup>38</sup> *Ibid* at 10.

constant from childhood into adulthood,<sup>39</sup> strongly indicating that, while our understanding of pain and the associations it elicits may become more complex over time, late-developing cortical circuitry is not required for a conscious experience of suffering.

35. The most scientifically accurate way of determining the neural structures required for a conscious experience of suffering (or any other conscious experience), independent of the activity of more basic brain regions that simply transmit pain information to our conscious awareness, is to directly stimulate a specific brain region in an alert patient and see if a pain response is elicited—thereby proving that the stimulated area is sufficient for a psychological experience of suffering. In agreement with decades of earlier research,<sup>40</sup> a recent study of over 4,000 stimulations of the cortex determined that pain responses were surprisingly rare (approximately 1.4%).<sup>41</sup> This demonstrates that, while the cortex may “process”

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<sup>39</sup> The Development of Nociceptive Circuits, Fitzgerald, M., *Nat. Rev. Neurosci.*, 2005 Jul; 6(7):507-20; Children's Ratings of Postoperative Pain Compared to Ratings by Nurses and Physicians, LaMontagne, L.L., Johnson, B.D., Hepworth, J.T., *Issues Compr. Pediatr. Nurs.*, 1991 Oct-Dec; 14(4):241-7; Management of Pain in Childhood, Harrop, J.E., *Arch. Dis. Child Educ. Pract. Ed.* 2007 Aug; 92(4):ep101-8.

<sup>40</sup> The Insula: Further Observations on its Function, Penfield, W., Faulk, M.E., Jr., *Brain*, 1955; 78(4):445-70; Penfield W., Jasper H., *Epilepsy and the Functional Anatomy of the Human Brain*, Boston: Brown, L., 1954.

<sup>41</sup> Stimulation of the Human Cortex and the Experience of Pain: Wilder Penfield's Observations Revisited, Mazzola, L., Isnard, J., Peyron, R., Mauguière, F., *Brain*, 2012; 135:631-40; See also Anatomofunctional Organization of the Insular Cortex: A Study Using Intracerebral Electrical Stimulation in Epileptic Patients, Afif, A., Minotti, L., Kahane, P., Hoffmann, D., *Epilepsia*, 2010 Nov; 51(11):2305-15; Pain-Related Neurons in the Human Cingulate Cortex, Hutchison, W.D., Davis, K.D., Lozano, A.M., Tasker, R.R., Dostrovsky, J.O., *Nat. Neurosci.*, 1999 May; 2(5):403-5;

painful experiences delivered from other brain regions, it is largely not involved in producing a conscious experience of pain, *i.e.* our conscious experience of suffering depends almost entirely on subcortical brain regions that develop very early in the human fetus.

36. Finally, a large body of direct experimental and medical evidence from adult humans afflicted with chronic pain contradicts the assertion of the RCOG and the JAMA that “suffering” requires cortical circuitry. Ablation<sup>42</sup> or stimulation<sup>43</sup> of the cortex does not affect pain perception, whereas altering the function of subcortical structures, including the thalamus, does.<sup>44</sup> Indeed, “Deep Brain Stimulation” of the

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Functional Mapping of the Insular Cortex: Clinical Implication in Temporal Lobe Epilepsy, Ostrowsky, K., Isnard, J., Ryvlin, P., Guénot, M., Fischer, C., Mauguière, F., *Epilepsia*, 2000 Jun; 41(6):681-6; Representation of Pain and Somatic Sensation in the Human Insula: A Study of Responses to Direct Electrical Cortical Stimulation, Ostrowsky, K., Magnin, M., Ryvlin, P., Isnard, J., Guénot, M., Mauguière, F., *Cereb. Cortex*, 2002 Apr; 12(4):376-85; Clinical Manifestations of Insular Lobe Seizures: A Stereo-Electroencephalographic Study, Isnard, J., Guénot, M., Sindou, M., Mauguière, F., *Epilepsia*, 2004 Sep; 45(9):1079-90.

<sup>42</sup> *Ibid* at 29.

<sup>43</sup> Motor Cortex Stimulation in Patients With Post-Stroke Pain: Conscious Somatosensory Response and Pain Control, Fukaya, C., Katayama, Y., Yamamoto, T., Kobayashi, K., Kasai, M., Oshima, H., *Neurol. Res.*, 2003; 25:153-6.; Stimulation of the Human Cortex and the Experience of Pain: Wilder Penfield’s Observations Revisited, Mazzola, L., Isnard, J., Peyron, R., Mauguière, F., *Brain*, 2012; 135:631-40.

<sup>44</sup> Somatotopic Organization of the Human Insula to Painful Heat Studied With High Resolution Functional Imaging, Brooks, J.C., Zambreau, L., Godinez, A., et al., *Neuroimage* 2005; 27:201-209; Thalamic Field Potentials in Chronic Central Pain Treated by Periventricular Gray Stimulation: A Series of Eight Cases, Nandi, D., Aziz, T., Carter, H., et al., *Pain*, 2003; 101:97-107; Thalamic Field Potentials During Deep Brain Stimulation of Periventricular Gray in Chronic Pain, Nandi, D., Liu, X., Joint, C., et al., *Pain*, 2002; 97:47- 51; Long-Term Outcomes of Deep Brain

thalamus, periaqueductal grey matter and internal capsule (all early developing, subcortical brain centers) has proven to be a highly effective treatment for chronic pain in human patients.<sup>45</sup>

37. Taken together, this extensive and diverse body of data clearly indicates that pain perception, including “suffering,” does not depend on cortical circuitry and is largely mediated by sub-cortical brain networks. And, as noted above, it is universally accepted that sub-cortical, spino-thalamic circuits capable of pain perception are established in a human fetus between 12-18 weeks.

38. What can we directly observe about how a fetus responds to painful stimuli? Multiple studies<sup>46</sup> clearly indicate that: “the human fetus from 18–20 weeks

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Stimulation For Neuropathic Pain, Boccard, S.G., Pereira, E.A., Moir, L., Aziz, T.Z., Green, A.L., *Neurosurgery*, 2013; 72:221-30; Regional Cerebral Perfusion Differences Between Periventricular Grey, Thalamic and Dual Target Deep Brain Stimulation For Chronic Neuropathic Pain, Pereira, E.A., Green, A.L., Bradley, K.M., Soper, N., Moir, L., Stein, J.F., Aziz, T.Z., *Stereotact Funct. Neurosurg.*, 2007; 85:175-83; Penfield, W., Jasper, H.H., *Epilepsy and the Functional Anatomy of the Human Brain*, Boston: Little, Brown & Co., 1954.

<sup>45</sup> Deep Brain Stimulation for Chronic Pain, Falowski, S.M., *Curr. Pain Headache Rep.* 2015 Jul; 19(7):27; Deep Brain Stimulation For Chronic Pain, Boccard, S.G., Pereira, E.A., Aziz, T.Z., *J. Clin. Neurosci.*, 2015 Jun 26. pii: S0967-5868(15)00218-0; Deep Brain Stimulation For Pain Relief: A Meta-Analysis, Bittar, R.G., Kar-Purkayastha, I., Owen, S.L., Bear, R.E., Green, A., Wang, S., Aziz, T.Z., *J. Clin. Neurosci.*, 2005 Jun; 12(5):515-9.

<sup>46</sup> The human fetus preferentially secretes corticosterone, rather than cortisol, in response to intrapartum stressors. Wynne-Edwards, K.E., Edwards H.E., Hancock, T.M., *PLoS One*, 2013 Jun 14; 8(6):e63684; Autonomous Adrenocorticotropin Reaction to Stress Stimuli in Human Fetus, Kosinska-Kaczynska, K., Bartkowiak, R., Kaczynski, B., Szymusik, I., Wielgos, M., *Early Hum. Dev.*, 2012 Apr; 88(4):197-201; Fetal Stress Response to Fetal Cardiac Surgery, Lam, C.T., Sharma, S., Baker, R.S., Hilshorst, J., Lombardi, J., Clark, K.E., Eghtesady, P., *Ann. Thorac. Surg.*, 2008 May; 85(5):1719-27; Human Fetal and Maternal Corticotrophin Releasing Hormone

elaborates pituitary-adrenal, sympatho-adrenal and circulatory stress responses to physical insults,” that can be eliminated by appropriate anesthesia.<sup>47</sup> In support of the conclusion that pain is experienced very early in human development, fetuses delivered prematurely (as early as 23 weeks) also show clear pain-related behaviors.<sup>48</sup> Strikingly, the earlier infants are delivered, the stronger their response to pain,<sup>49</sup> perhaps due to the absence of late developing cortical circuits that inhibit pain perception.<sup>50</sup>

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Responses to Acute Stress, Gitau, R., Fisk, N.M., Glover, V., Arch. Dis. Child. Fetal Neonatal Ed., 2004 Jan; 89(1):F29-32; Fetal hypothalamic-pituitary-adrenal stress responses to invasive procedures are independent of maternal responses. Gitau, R., Fisk, N.M., Teixeira, J.M., Cameron, A., Glover, V. J., Clin. Endocrinol Metab., 2001 Jan; 86(1):104-9; Acute Cerebral Redistribution in Response to Invasive Procedures in the Human Fetus, Teixeira, J.M., Glover, V., Fisk, N.M., Am. J. Obstet. Gynecol., 1999 Oct; 181(4):1018-25; Fetal Plasma Cortisol and Beta-Endorphin Response to Intrauterine Needling, Giannakouloupoulos, X., Sepulveda, W., Kourtis, P., Glover, V., Fisk, N.M., Lancet. 1994 Jul 9;344(8915):77-81; Acute Increase In Femoral Artery Resistance In Response to Direct Physical Stimuli In the Human Fetus, Smith, R.P., Glover, V., Fisk, N.M., BJOG, 2003 Oct; 110(10):916-21.

<sup>47</sup> Effect of Direct Fetal Opioid Analgesia on Fetal Hormonal and Hemodynamic Stress Response to Intrauterine Needling, Fisk, N.M., Gitau, R., Teixeira, J.M., Giannakouloupoulos, X., Cameron, A.D., Glover, V.A., Anesthesiology, 2001;95:828-35.

<sup>48</sup> Pain Behaviours in Extremely Low Gestational Age Infants, Gibbins, S., Stevens, B., Beyene, J., Chan, P.C., Bagg, M., Asztalos, E., Early Hum. Dev., 2008;84:451-8.

<sup>49</sup> Determinants of Premature Infant Pain Responses to Heel Sticks, Badr, L.K., Abdallah, B., Hawari, M., Sidani, S., Kassir, M., Nakad, P., Breidi, J., Pediatr. Nurs., 2010;36:129-36.

<sup>50</sup> Descending Pain Modulation and Chronification of Pain, Ossipov, M.H., Morimura, K., Porreca, F., Curr. Opin. Support Palliat Care, 2014 Jun; 8(2):143-51; The Role of Descending Inhibitory Pathways on Chronic Pain Modulation and Clinical Implications, Kwon, M., Altin, M., Duenas, H., Alev, L., Pain Pract., 2014 Sep; 14(7):656-67; The Consequences of Pain in Early Life: Injury-Induced Plasticity in

39. Finally, painful experiences during prenatal life can potentially have long-term impact on neural development,<sup>51</sup> with one pain expert stating: “Whereas evidence for conscious pain perception is indirect, evidence for the subconscious incorporation of pain into neurological development and plasticity is incontrovertible.”<sup>52</sup>

40. These and many other direct observations of fetal behavior and physiology have resulted in a clear consensus among professional anesthesiologists that the use of medications to relieve pain is warranted in cases of fetal surgery.<sup>53</sup> Many of the advocates of fetal anesthesia make no claims regarding the qualitative

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Developing Pain Pathways, Schwaller, F., Fitzgerald, M., *Eur. J. Neurosci.*, 2014 Feb; 39(3):344-52.

<sup>51</sup> Pain and Stress in the Human Fetus, Smith, R.P., Gitau, R., Glover, V., Fisk, N.M., *Eur. J. Obstet. Gynecol. Reprod. Biol.* 2000 Sep; 92(1):161-5; Pain and Stress in the Human Fetus, White, M.C., Wolf, A.R., *Best Pract. Res. Clin. Anaesthesiol.*, 2004 June 18(2):205-20; Management of Fetal Pain During Invasive Fetal Procedures: A Review, Huang, W., Deprest, J., Missant, C., Van de Velde, M., *Acta Anaesthesiol. Belg.* 2004;55(2):119-23.

<sup>52</sup> Neurodevelopmental Changes of Fetal Pain, Lowery, C.L., Hardman, M.P., Manning, N., Hall, R.W., Anand, K.J., Clancy, B., *Semin. Perinatol.*, 2007 Oct; 31(5):275-82.

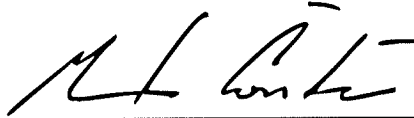
<sup>53</sup> Use of Fetal Analgesia During Prenatal Surgery, Bellieni, C.V., Tei, M., Stazzoni, G., Bertrando, S., Cornacchione, S., Buonocore, G.J., *Matern. Fetal Neonatal Med.* 2013; 26:90-5; Towards State-of-the-Art Anesthesia for Fetal Surgery: Obstacles and Opportunities, Kuczkowski, K.M., *Rev. Esp. Anesthesiol Reanim*, 2013 Jan; 60(1):3-6; Fetal and Maternal Analgesia/Anesthesia for Fetal Procedures, Van de Velde, M., De Buck, F., *Fetal Diagn. Ther.*, 2012; 31:201-9; Anesthesia for Fetal Surgery, Lin, E.E., Tran, K.M., *Semin. Pediatr. Surg.*, 2013; 22:50-5; Anesthesia for in Utero Repair of Myelomeningocele, Ferschl, M., Ball, R., Lee, H., Rollins, M.D., *Anesthesiology*, 2013; 118:1211-23; Anesthesia for Fetal Surgery, Tran, K.M., *Semin. Fetal Neonatal Med.*, 2010 Feb; 15(1):40-5; Anesthesia for Fetal Procedures and Surgery, Rosen, M.A., *Yonsei Med. J.*, 2001 Dec; 42(6):669-80.

nature of fetal pain, but, based on both the scientific literature and on their own observations, they clearly conclude that pain exists for these fetuses and that they are obligated to address fetal pain medically, despite the many serious challenges and medical risks entailed in providing pain relief to a fetus in utero.

41. I am not being compensated financially in any way for my testimony given in this Declaration.

42. My fee for giving any deposition(s) in cases such as this is \$1,000.00 per hour for testimonial time, \$75.00 per hour for travel time, \$400.00 per hour for time spent reviewing and correcting any resulting deposition transcript and 100% of any out-of-pocket expenses incurred.

I declare under penalty of perjury that the foregoing is true and correct to the best of my knowledge.



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Maureen L. Condic

Dated: 27, November, 2017

CERTIFICATE OF SERVICE

This is to certify that, on the 31<sup>st</sup> day of July 2017, I provided a notice copy of the foregoing Declaration to counsel for the plaintiffs, as follows:

Christopher Brook, Esquire  
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BY FIRST-CLASS U.S. MAIL AND  
BY ELECTRONIC MAIL

This is to further certify that, on the 4<sup>th</sup> day of December 2017, I electronically filed the foregoing Declaration with the Clerk of Court, using the CM/ECF system, which will send a notice of electronic filing to all counsel of record who have appeared in this case.

/S/ Isham Faison Hicks  
Isham Faison Hicks  
*Attorney for the Defendants*